

☐ 1: Virology. 1996 Mar 15;217(2):594-7.

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Disassembly of the coliphage lambda replication complex due to heat shock induction of the groE operon.

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We have found previously that, in contrast to the free O initiator protein of lambda phage or plasmid rapidly degraded by the Escherichia coli ClpP/ClpX protease, the lambda O present in the replication complex (RC) is protected from proteolysis. In amino acid-starved E. coli relA cells, a temperature shift from 30 to 43 degrees did not affect RC integrity, as judged from the unchanged level of stable lambda O observed; however, the same temperature shift in a complete medium resulted in the decay of this lambda O fraction, which suggested disassembly of the RC. Examination of this phenomenon revealed that for lambda RC disassembly, heat shock induction of the groE operon, coding for molecular chaperones of the Hsp60 class, is indispensable. Heat shock induction of the groE operon present on a multicopy plasmid inhibited the growth of infecting phage.

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Mol Microbiol. 1997 May;24(4):687-95.

Links

Structural analysis of the subunits of the trehalose-6-phosphate synthase/phosphatase complex in *Saccharomyces cerevisiae* and their function during heat shock.

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Synthesis of trehalose in the yeast *Saccharomyces cerevisiae* is catalysed by the trehalose-6-phosphate (Tre6P) synthase/phosphatase complex, which is composed of at least three different subunits encoded by the genes TPS1, TPS2, and TSL1. Previous studies indicated that Tps1 and Tps2 carry the catalytic activities of trehalose synthesis, namely Tre6P synthase (Tps1) and Tre6P phosphatase (Tps2), while Tsl1 was suggested to have regulatory functions. In this study two different approaches have been used to clarify the molecular composition of the trehalose synthase complex as well as the functional role of its potential subunits. Two-hybrid analyses of the *in vivo* interactions of Tps1, Tps2, Tsl1, and Tps3, a protein with high homology to Tsl1, revealed that both Tsl1 and Tps3 can interact with Tps1 and Tps2; the latter two proteins also interact with each other. In addition, trehalose metabolism upon heat shock was analysed in a set of 16 isogenic yeast strains carrying deletions of TPS1, TPS2, TSL1, and TPS3 in all possible combinations. These results not only confirm the previously suggested roles for Tps1 and Tps2, but also provide, for the first time, evidence that Tsl1 and Tps3 may share a common function with respect to regulation and/or structural stabilization of the Tre6P synthase/phosphatase complex in exponentially growing, heat-shocked cells.

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[Links](#)

Heat shock induction by a misassembled cytoplasmic membrane protein complex in *Escherichia coli*.

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We analysed the effects of the overproduction of parts or all of a multisubunit ATP-binding cassette (ABC) transporter, the MalFGK2 complex, involved in the uptake of maltose and maltodextrins in *Escherichia coli*. We found that production of the MalF protein alone was inducing the *phtrA* promoter, which is under the control of a recently discovered sigma factor, sigma24, involved in the response to extracytoplasmic stresses. The production level, stability and localization of MalF were not altered when produced without its partners, suggesting that the protein was correctly inserted in the membrane. Our results indicate that a large periplasmic loop located between the third and fourth transmembrane segment of MalF, the L3 loop, is responsible for *phtrA* induction: (i) deleted MalF proteins with no L3 loop or with a L3 loop lacking 120 amino acids do not induce the *phtrA* promoter; (ii) the export to the periplasm of the L3 loop alone or fused to MalE induces the *phtrA* promoter. Moreover, the proteolytic sensitivity of MalF is different when it is produced alone and when MalF and MalG are produced together, suggesting a change in the conformation and/or accessibility of MalF. These results suggest that some inner membrane proteins can be sensed outside the cytoplasm by a quality control apparatus or by the export machinery. Moreover, the observation of the *phtrA* induction by MalF could be a useful new tool for studying the insertion and assembly of the MalFGK2 complex.

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